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NEWS 30 Oct 25 MEDLINE SDI run of October 8, 2002
NEWS 31 Nov 18 DKILIT has been renamed APOLLIT
NEWS 32 Nov 25 More calculated properties added to REGISTRY
NEWS 33 Dec 02 TIBKAT will be removed from STN
NEWS 34 Dec 04 CSA files on STN
NEWS 35 Dec 17 PCTFULL now covers WPIPCT Applications from 1978 to date
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L4 ANSWER 1 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE
1
AN 2002:535460 BIOSIS
DN PREV200200535460
TI cis elements of the villin gene control expression in restricted domains of the vertical (crypt) and horizontal (duodenum, cecum) axes of the intestine.
AU Madison, Blair B.; Dunbar, Laura; Qiao, Xiaotan T.; Braunstein, Katherine; Braunstein, Evan; Gumucio, Deborah L. (1)
CS (1) Dept. of Cell and Developmental Biology, University of Michigan Medical School, 5704 Medical Science II, Ann Arbor, MI, 48109-0616: dgumucio@umich.edu USA
SO Journal of Biological Chemistry, (September 6, 2002) Vol. 277, No. 36, pp. 33275-33283. http://www.jbc.org/. print.
ISSN: 0021-9258.
DT Article
LA English
AB ***Villin*** , an actin bundling protein found in the apical brush border of absorptive tissues, is one of the first structural genes to be transcriptionally activated in the embryonic intestinal endoderm. In the adult, ***villin*** is broadly expressed in every cell of the intestinal epithelium on both the vertical axis (crypt to villus tip) and the horizontal axis (duodenum through colon) of the intestine. Here, we document that a 12.4-kilobase region of the ***mouse*** ***villin*** gene drives high level expression of two different reporter genes (LacZ and Cre recombinase) within the entire intestinal epithelium of transgenic ***mice*** . Deletion of a portion of this transgene results in reduction of beta-galactosidase activity in restricted domains of the small intestine (duodenum) and large intestine (cecum). In addition, expression is reduced in the crypt compartment throughout the intestine. Thus, the global expression pattern of ***villin*** in the intestine is apparently the consequence of an amalgam of distinct and individual domain-specific control processes. That is, expression of ***villin*** in the duodenum and cecum requires different ***regulatory*** sequences than the rest of the intestine, and the expression of ***villin*** in crypts is ***regulated*** by different circuitry than expression of ***villin*** on villus tips.

L4 ANSWER 2 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE
2
AN 2002:340376 BIOSIS
DN PREV200200340376
TI Headpiece domain of dematin is required for the stability of the erythrocyte membrane.
AU Khanna, Richie; Chang, Seon H.; Andrabi, Shaida; Azam, Mohammad; Kim, Anthony; Rivera, Alicia; Brugnara, Carlo; Low, Philip S.; Liu, Shih-Chun; Chishti, Athar H. (1)
CS (1) Departments of Medicine, Anatomy, and Cellular Biology, St. Elizabeth's Medical Center, Tufts University School of Medicine, Boston, MA, 02135: athar.chishti@tufts.edu USA
SO Proceedings of the National Academy of Sciences of the United States of America, (May 14, 2002) Vol. 99, No. 10, pp. 6637-6642. http://www.pnas.org. print.
ISSN: 0027-8424.
DT Article
LA English
AB Dematin is an actin-binding and bundling protein of the erythrocyte membrane skeleton. Dematin is localized to the spectrin-actin junctions, and its actin-bundling activity is ***regulated*** by phosphorylation of cAMP-dependent protein kinase. The carboxyl terminus of dematin is homologous to the "headpiece" domain of ***villin*** , an actin-bundling protein of the microvillus cytoskeleton. The headpiece domain contains an actin-binding site, a cAMP-kinase phosphorylation site, plays an essential role in dematin self-assembly, and bundles F-actin in vitro. By using homologous recombination in ***mouse*** embryonic stem cells, the headpiece domain of dematin was deleted to evaluate its

function in vivo. Dematin headpiece null ***mice*** were viable and born at the expected Mendelian ratio. Hematological evaluation revealed evidence of compensated anemia and spherocytosis in the dematin headpiece null ***mice*** . The headpiece null erythrocytes were osmotically fragile, and ektacytometry/micropore filtration measurements demonstrated reduced deformability and filterability. In vitro membrane stability measurements indicated significantly greater membrane fragmentation of the dematin headpiece null erythrocytes. Finally, biochemical characterization, including the vesicle/cytoskeleton dissociation, spectrin self-association, and chemical crosslinking measurements, revealed a weakened membrane skeleton evidenced by reduced association of spectrin and actin to the plasma membrane. Together, these results provide evidence for the physiological significance of dematin and demonstrate a role for the headpiece domain in the maintenance of structural integrity and mechanical properties of erythrocytes in vivo.

L4 ANSWER 3 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE
3
AN 2002:298270 BIOSIS
DN PREV200200298270
TI Fliih, a gelsolin-related cytoskeletal regulator essential for early mammalian embryonic development.
AU Campbell, Hugh D. (1); Fountain, Shelley; McLennan, Ian S.; Berven, Leise A.; Crouch, Michael F.; Davy, Deborah A.; Hooper, Jane A.; Waterford, Kynan; Chen, Ken-Shiung; Lupski, James R.; Ledermann, Birgit; Young, Ian G.; Matthaei, Klaus I.
CS (1) Molecular Genetics and Evolution Group, Research School of Biological Sciences, Australian National University, Canberra, ACT, 2601: Hugh.Campbell@anu.edu.au Australia
SO Molecular and Cellular Biology, (May, 2002) Vol. 22, No. 10, pp. 3518-3526. http://mcb.asm.org/. print.
ISSN: 0270-7306.
DT Article
LA English
AB The Drosophila melanogaster flightless I gene is required for normal cellularization of the syncytial blastoderm. Highly conserved homologues of flightless I are present in Caenorhabditis elegans, ***mouse*** , and human. We have disrupted the ***mouse*** homologue Fliih by homologous recombination in embryonic stem cells. Heterozygous Fliih mutant ***mice*** develop normally, although the level of Fliih protein is reduced. Cultured homozygous Fliih mutant blastocysts hatch, attach, and form an outgrowing trophoblast cell layer, but egg cylinder formation fails and the embryos degenerate. Similarly, Fliih mutant embryos initiate implantation in vivo but then rapidly degenerate. We have constructed a transgenic ***mouse*** carrying the complete human FLII gene and shown that the FLII transgene is capable of rescuing the embryonic lethality of the homozygous targeted Fliih mutation. These results confirm the specific inactivation of the Fliih gene and establish that the human FLII gene and its gene product are functional in the ***mouse*** . The Fliih ***mouse*** mutant phenotype is much more severe than in the case of the related gelsolin family members gelsolin, ***villin*** , and CapG, where the homozygous mutant ***mice*** are viable and fertile but display alterations in cytoskeletal actin ***regulation*** .

L4 ANSWER 4 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE
4
AN 2002:441543 BIOSIS
DN PREV200200441543
TI Targeted expression of oncogenic K-ras in intestinal epithelium causes spontaneous tumorigenesis in mice.
AU Janssen, Klaus-Peter; El Marjou, Fatima; Pinto, Daniel; Sastre, Xavier; Rouillard, Dany; Fouquet, Coralie; Soussi, Thierry; Louvard, Daniel; Robine, Sylvie (1)
CS (1) CNRS-UMR144, Institut Curie, 26 Rue d'Ulm, 75248, Paris Cedex 05: sylvie.robine@curie.fr France
SO Gastroenterology, (August, 2002) Vol. 123, No. 2, pp. 492-504. http://www.gastrojournal.org/. print.
ISSN: 0016-5085.
DT Article
LA English
AB Background & Aims: Ras oncoproteins are mutated in about 50% of human colorectal cancers, but their precise role in tumor initiation or progression is still unclear. Methods: This study presents transgenic ***mice*** that express K-rasV12G, the most frequent oncogenic mutation in human tumors, under control of the ***murine*** ***villin*** ***promoter*** in epithelial cells of the large and small intestine. Results: More than 80% of the transgenic animals displayed single or multiple intestinal lesions, ranging from aberrant crypt foci (ACF) to invasive adenocarcinomas. Expression of K-rasV12G caused activation of the MAP kinase cascade, and the tumors were frequently characterized by deregulated cellular proliferation. Unexpectedly, we obtained no evidence of inactivating mutations of the tumor suppressor gene Apc, the "gatekeeper" in colonic epithelial proliferation. However, spontaneous mutation of the tumor-suppressor gene p53, a frequent feature in the human disease, was found in 3 of 7 tumors that were tested. Conclusions: This animal model recapitulates the stages of tumor progression as well as a part of the genetic alterations found in human colorectal cancer. Furthermore, it indicates that activation of K-ras in concert with mutations in p53 may constitute a route to digestive tumor formation and growth, underlining the fact that the pathway to intestinal cancer is not necessarily a single road.

L4 ANSWER 5 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE
5
AN 2001:441295 BIOSIS
DN PREV200100441295
TI Comparisons of CapG and gelsolin-null macrophages: Demonstration of a unique role for CapG in receptor-mediated ruffling, phagocytosis, and vesicle rocketing.
AU Witke, Walter; Li, Wei; Kwiatkowski, David J. (1); Southwick, Frederick S.
CS (1) Division of Experimental Medicine, Brigham and Women's Hospital, Harvard Medical School, 221 Longwood Ave., Boston, MA, 02115: dkwiatkowski@rics.bwh.harvard.edu USA
SO Journal of Cell Biology, (August 20, 2001) Vol. 154, No. 4, pp. 775-784. print.
ISSN: 0021-9525.
DT Article
LA English
SL English
AB Capping the barbed ends of actin filaments is a critical step for ***regulating*** actin-based motility in nonmuscle cells. The in vivo function of CapG, a calcium-sensitive barbed end capping protein and member of the gelsolin/ ***villin*** family, has been assessed using a null Capg allele engineered into ***mice*** . Both CapG-null ***mice*** and CapG/gelsolin double-null ***mice*** appear normal and have no gross functional abnormalities. However, the loss of CapG in bone marrow macrophages profoundly inhibits macrophage colony stimulating factor-stimulated ruffling; reintroduction of CapG protein by microinjection fully restores this function. CapG-null macrophages also demonstrate apprx50% impairment of immunoglobulin G, and complement-opsonized phagocytosis and lanthanum-induced vesicle rocketing. These motile functions are not impaired in gelsolin-null macrophages and no additive effects are observed in CapG/gelsolin double-null macrophages, establishing that CapG function is distinct from, and does not overlap with, gelsolin in macrophages. Our observations indicate that CapG is required for receptor-mediated ruffling, and that it is a major functional component of macrophage phagocytosis. These primary effects on macrophage motile function suggest that CapG may be a useful target for the ***regulation*** of macrophage-mediated inflammatory responses.

L4 ANSWER 6 OF 22 CAPLUS COPYRIGHT 2003 ACS
AN 2000:402016 CAPLUS
DN 133:54573
TI ***Regulatory*** sequences of the ***mouse*** ***villin*** gene and the therapeutic uses thereof
IN Pinto, Daniel; Robine, Sylvie; Jaisser, Frederic; Louvard, Daniel
PA Centre National de la Recherche Scientifique, Fr.; Institut Curie
SO PCT Int. Appl., 54 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000034492	A1	20000615	WO 1998-EP8009	19981209
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
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AU 9922692	A1	20000626	AU 1999-22692	19981209
WO 2000034493	A2	20000615	WO 1999-EP9782	19991209
WO 2000034493	A3	20001116		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1137791	A2	20011004	EP 1999-963487	19991209
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
US 2002102705	A1	20020801	US 2001-877935	20010608
PRAI WO 1998-EP8009	A	19981209		
WO 1999-EP9782	W	19991209		

AB The invention relates to ***regulatory*** sequences of the ***mouse*** ***villin*** gene that efficiently drive transgenic expression in immature and differentiated epithelial cells of the intestine and urogenital tracts. The invention also relates to recombinant constructs comprising ***mouse*** ***villin*** gene ***regulatory*** sequences, for the control of the targeted tissue-specific or cell-specific expression of detd. DNA sequences such as genes with therapeutic values and genes used to study the mol. and cellular basis of normal and pathol. states, in cells or tissues originating from the intestinal mucosa. The invention further provides cells, tissues or organisms including animals, expressing detd. DNA

sequences in a targeted manner.
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L4 ANSWER 7 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 2001:314512 BIOSIS
DN PREV200100314512
TI Knocking out CapG, a calcium-sensitive actin filament barbed end capping protein, profoundly impairs macrophage receptor mediated ruffling, phagocytosis and endosomal rocketing.
AU Southwick, Frederick S. (1); Witke, Walter; Li, Wei (1); Kwiatkowski, David J.
CS (1) Medicine, University of Florida, Gainesville, FL USA
SO Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 444a. print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000 American Society of Hematology
. ISSN: 0006-4971.
DT Conference
LA English
SL English
AB Capping the barbed ends of actin filaments is a critical step for ***regulating*** actin-based motility in nonmuscle cells. We have examined the in vivo function of CapG, a calcium-sensitive barbed end capping protein and member of the gelsolin- ***villin*** family, by creating a null Capg allele engineered in ***mice*** . Loss of CapG profoundly inhibited macrophage ruffling. As assessed by rhodamine phalloidin staining, spontaneous macrophage ruffling was markedly reduced (ruffling index: null cells, 0.19 +/- 0.02 SEM vs. wild-type, 0.50 +/- 0.02, n = 94), and null macrophages failed to respond to MCSF (null cells post MCSF, 0.23 +/- 0.02 vs wild-type post MCSF, 0.84 +/- 0.07, n = 100). Introduction of CapG by microinjection (needle conc. 30 mg/ml) into null cells caused a recovery of MCSF responsiveness (post MCSF ruffling index of injected cells: 1.10 +/- 0.1 vs uninjected cells 0.23 +/- 0.02, n = 34). Loss of CapG also had a marked inhibitory effect on phagocytosis. The phagocytic rates of IgG and complement opsonized zymosan particles in CapG null macrophages were reduced by apprxeq1/2 (Examples: IgG opsonized zymosan: 22 min, null = 3.8 +/- 0.3 vs wild-type = 7.2 +/- 0.7 particles/cell, n = 100; Complement opsonized zymosan: 15 min, null = 5.1 +/- 0.3 vs wild-type = 8.0 +/- 0.3 particles/cell, n = 100). Finally the velocity of lanthanum-induced endosomal rocketing (an actin-based motility test developed in our laboratory) was reduced by 50% in CapG null macrophages (0.03 +/- - 0.002 vs wild-type, 0.06 +/- 0.002 um/sec, n = 100). CapG function was distinctly different from gelsolin as evidenced by a lack of inhibition of these motile functions in gelsolin null macrophages and the absence of any additive effects by breeding gelsolin-CapG double knock-out ***mice*** . We conclude that CapG serves unique actin- ***regulatory*** functions and is a major component of receptor mediated ruffling and phagocytosis in macrophages. These primary effects on macrophage motile function suggest that CapG may be a useful target for ***regulating*** macrophage mediated inflammatory responses.

L4 ANSWER 8 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE
6
AN 1999:195094 BIOSIS
DN PREV199900195094
TI ***Regulatory*** sequences of the ***mouse*** ***villin*** gene that efficiently drive transgenic expression in immature and differentiated epithelial cells of small and large intestines.
AU Pinto, Daniel; Robine, Sylvie; Jaisser, Frederic; Marjou, Fatima El; Louvard, Daniel (1)
CS (1) UMR 144 CNRS, Institut Curie, 26 rue d'Ulm, 75248 Paris Cedex 05 France
SO Journal of Biological Chemistry, (March 5, 1999) Vol. 274, No. 10, pp. 6476-6482.
ISSN: 0021-9258.
DT Article
LA English
AB ***Villin*** is an early marker of epithelial cells from the digestive and urogenital tracts. Indeed ***villin*** is expressed in the stem cells and the proliferative cells of the intestinal crypts. To investigate the underlying molecular mechanisms and particularly those responsible for the restricted tissue specificity, a large genomic region of the ***mouse*** ***villin*** gene has been analyzed. A 9-kilobase (kb) ***regulatory*** region of the ***mouse*** ***villin*** gene (harboring 3.5 kb upstream the transcription start site and 5.5 kb of the first intron) was able to promote transcription of the LacZ reporter gene in the small and large intestines of transgenic ***mice*** , in a transmissible manner, and thus efficiently directed subsequent beta-galactosidase expression in epithelial cells along the entire crypt-villus axis. In the kidney, the transgene was also expressed in the epithelial cells of the proximal tubules but is likely sensitive to the site of integration. A construct lacking the first intron restricted beta-galactosidase expression to the small intestine. Thus, the 9-kb genomic region contains the necessary cis-acting elements to recapitulate the tissue-specific expression pattern of the endogenous ***villin*** gene. Hence, these ***regulatory*** sequences can be used to target heterologous genes in immature and differentiated epithelial cells of the small and/or large intestinal mucosa.

L4 ANSWER 9 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE
7
AN 1998:428551 BIOSIS
DN PREV199800428551
TI Advillin (p92): A new member of the gelsolin/villin family of actin regulatory proteins.
AU Marks, Peter W. (1); Arai, Maya; Bandura, Jennifer L.; Kwiatkowski, David J.
CS (1) Div. Exp. Med., Dep. Med., Brigham Women's Hosp., Harvard Med. Sch., Boston, MA USA
SO Journal of Cell Science, (Aug., 1998) Vol. 111, No. 15, pp. 2129-2136.
ISSN: 0021-9533.
DT Article
LA English
AB A new member of the gelsolin/ ***villin*** family of actin ***regulatory*** proteins was initially identified by screening an adult ***murine*** brain cDNA library with a probe for bovine adseverin. The predicted amino acid sequence of the 92 kDa ***murine*** protein p92 (advillin) is 75% homologous to ***villin*** and 65% homologous to gelsolin and adseverin. It shares a six domain structure with other gelsolin family members and has a carboxy-terminal headpiece, similar to, yet distinct from, ***villin*** . Northern blot analysis shows a high level of mRNA expression in ***murine*** uterus and human intestine. In situ mRNA analysis of adult ***murine*** tissues demonstrates that the message is most highly expressed in the endometrium of the uterus, the intestinal lining, and at the surface of the tongue. In ***murine*** embryonic development, strong expression of the message is observed by day 14.5 in dorsal root ganglia and trigeminal ganglia. Expression is also noted at day 16.5 in cerebral cortex. We propose that p92 (advillin) has unique functions in the morphogenesis of neuronal cells which form ganglia, and that it may compensate to explain the near normal phenotype observed in ***villin*** -deficient ***mice*** .

L4 ANSWER 10 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE
8
AN 1999:72231 BIOSIS
DN PREV199900072231
TI Cytoskeletal reorganization by soluble Wnt-3a protein signalling.
AU Shibamoto, Sayumi; Higano, Keiichi; Takada, Ritsuko; Ito, Fumiaki; Takeichi, Masatoshi; Takada, Shinji (1)
CS (1) Center Molecular and Developmental Biol., Kyoto Univ., Kitashirakawa, Sakyo-ku, Kyoto 606-8502 Japan
SO Genes to Cells, (Dec., 1998) Vol. 3, No. 10, pp. 659-670.
ISSN: 1356-9597.
DT Article
LA English
AB Background: Wnt-3a is an intercellular signalling molecule that is involved in a variety of morphogenetic events. However, the molecular mechanisms underlying Wnt-3a signalling are poorly understood. We have sought to establish in vitro systems to assay the activity of this protein and investigate its biological roles. Results: We prepared ***mouse*** L cells transfected with Wnt-3a cDNA, and found that their beta-catenin protein level was up- ***regulated*** . When conditioned medium (CM) was collected from cultures of the transfectants and added to nontransfected L cells, the beta-catenin level of the latter was also increased. Approximately 50% of the Wnt-3a proteins synthesized by the transfectants were secreted into the CM in a soluble form. These secreted Wnt-3a proteins formed an activity gradient in the environment surrounding the transfectants. Then, we studied whether Wnt-3a had any effect on cellular behaviour in vitro. When the CM containing Wnt-3a (W3a-CM) was added to cultures of C57MG mammary epithelial cells, their morphology was altered to exhibit closer intercellular contacts. Immunostaining for various adhesion and cytoskeletal proteins showed that the actin-microfilamental system was re-organized by the W3a-CM treatment. It induced a directional alignment of actin stress fibres and other actin-associated proteins. Moreover, ***villin*** , localized only at the perinuclear regions in untreated C57MG cells, was re-distributed to the leading edges of the cells, co-localizing with F-actin, in the presence of Wnt-3a. Conclusion: Our findings suggest that Wnt-3a protein, in the soluble form, can act to re-organize cytoskeletal structures.

L4 ANSWER 11 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1999:17581 BIOSIS
DN PREV199900017581
TI Functional analysis of the ***mouse*** ***villin*** gene ***promoter*** .
AU Dunbar, L.; Yu, T.; Braunstein, E.; Gumucio, D.
CS Dep. Anatomy Cell Biol., Univ. Mich., Ann Arbor, MI 48109 USA
SO Molecular Biology of the Cell, (Nov., 1998) Vol. 9, No. SUPPL., pp. 317A.
Meeting Info.: 38th Annual Meeting of the American Society for Cell Biology San Francisco, California, USA December 12-16, 1998 American Society for Cell Biology
. ISSN: 1059-1524.
DT Conference
LA English

L4 ANSWER 12 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE
9

AN 1997:156163 BIOSIS
DN PREV199799455366
TI E2a-Pbx1 induces aberrant expression of tissue-specific and developmentally regulated genes when expressed in NIH 3T3 fibroblasts.
AU Fu, Xinyu (1); Kamps, Mark P.
CS (1) Dep. Pathol., Univ. California, San Diego, Sch. Med., 9500 Gilman Dr., La Jolla, CA 92093 USA
SO Molecular and Cellular Biology, (1997) Vol. 17, No. 3, pp. 1503-1512. ISSN: 0270-7306.
DT Article
LA English
AB The E2a-Pbx1 oncoprotein contains the transactivation domain of E2a joined to the DNA-binding homeodomain (HD) of Pbx1. In ***mice*** , E2a-Pbx1 transforms T lymphoblasts and fibroblasts and blocks myeloblast differentiation. Pbx1 and E2a-Pbx1 bind DNA as heterodimers with other HD proteins whose expression is tissue specific. While the transactivation domain of E2a is required for all forms of transformation, DNA binding by the Pbx1 HD is essential for blocking myeloblast differentiation but dispensable for fibroblast or T-lymphoblast transformation. These properties suggest (i) that E2a-Pbx1 causes cellular transformation by activating gene transcription, (ii) that transcription of E2a-Pbx1 target genes is normally ***regulated*** by ubiquitous Pbx proteins and tissue-specific partners, and (iii) that DNA-binding mutants of E2a-Pbx1 activate a subset of all gene targets. To test these predictions, genes induced in NIH 3T3 fibroblasts by E2a-Pbx1 were identified and examined for tissue- and stage-specific expression and their differential abilities to be upregulated by E2a-Pbx1 in NIH 3T3 fibroblasts and myeloblasts and by a DNA-binding mutant of E2a-Pbx1 in NIH 3T3 cells. Of 12 RNAs induced by E2a-Pbx1, 4 encoded known proteins (a J-C region of the immunoglobulin kappa light chain, natriuretic peptide receptor C, mitochondrial fumarase, and the 3',5'-cyclic nucleotide phosphodiesterase, PDE1A) and 5 encoded new proteins related to angiogenin, ion channels, ***villin*** , epidermal growth factor repeat proteins, and the human 2.19 gene product. Expression of many of these genes was tissue specific or developmentally ***regulated*** , and most were not expressed in fibroblasts, indicating that E2a-Pbx1 can induce ectopic expression of genes associated with lineage-specific differentiation.

L4 ANSWER 13 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 10
AN 1997:405302 BIOSIS
DN PREV199799711505
TI Activity and inducibility of drug-metabolizing enzymes in immortalized hepatocyte-like cells (mhPKT) derived from a L-PK/Tag1 transgenic mouse.
AU Courjault-Gautier, Francoise; Antoine, Benedicte; Bens, Marcelle; Vallet, Veronique; Cluzeaud, Francoise; Pringault, Eric; Kahn, Axel; Toutain, Herve; Vandewalle, Alain (1)
CS (1) Faculte Med. Xavier Bichat, Inst. Federatif Recherche 02, INSERM U246, B.P. 416, 75870 Paris France
SO Experimental Cell Research, (1997) Vol. 234, No. 2, pp. 362-372. ISSN: 0014-4827.
DT Article
LA English
AB This report describes the establishment and characterization of the mhPKT cell line derived from the liver of a transgenic ***mouse*** harboring the simian virus (SV40) large T and small t antigens placed under the control of the 5' ***regulatory*** sequence of the rat L-type pyruvate kinase (L-PK) gene. mhPKT cells had a prolonged life span, expressed the SV40-encoded nuclear large T antigen when grown in glucose-enriched medium, and induced tumors when injected subcutaneously into athymic (nu-nu) ***mice*** . Growth on petri dishes or filters yielded multiple layers of cuboid cells, with numerous spaces between adjacent cells that were closed by junctional complexes. These bile canaliculi-like structures exhibited numerous microvilli in which ***villin*** , an actin-binding brush-border protein, colocalized with actin. These bile canaliculi-like structures appeared to be functional as they accumulated fluorescein. mhPKT cells conserved the expression of the liver-specific transcription factors HNF1, HNF3, HNF4, and DBP together with substantial levels of L-PK and albumin but not alpha-fetoprotein mRNA transcripts. mhPKT cells mainly metabolized testosterone into androstenedione and 6-beta-hydroxytestosterone, as in vivo. 3-Methylcholanthrene and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) markedly increased ethoxyresorufin-O-deethylase activity and the related cytochrome P450 (CYP) 1A1/2 protein, whereas alpha-naphthoflavone antagonized the TCDD-elicited induction. Phenobarbital slightly increased the CYP2B-mediated activities of pentoxyresorufin-O-dependentylase, 2-beta- and 16-beta-testosterone hydroxylase. mhPKT cells also had substantial sulfotransferase, UDP-glucuronyltransferase, and glutathione Stransferase activities. This model may serve as a tool for long-term in vitro studies of xenobiotic metabolism, potent CYP inducers, and hepatocyte damage due to drugs and other factors.

L4 ANSWER 14 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 11
AN 1996:411958 BIOSIS
DN PREV199699134314
TI Transimmortalized mouse intestinal cells (m-IC-cl2) that maintain a crypt phenotype.
AU Bens, Marcelle; Bogdanova, Anna; Cluzeaud, Francoise; Miquerol, Lucille; Kerneis, Sophie; Kraehenbuhl, Jean Pierre; Kahn, Axel; Pringault, Eric; Vandewalle, Alain (1)
CS (1) INSERM U246, Faculte de Medecine Xavier Bichat, B.P.416, 75870 Paris Cedex 18 France
SO American Journal of Physiology, (1996) Vol. 270, No. 6 PART 1, pp. C1666-C1674. ISSN: 0002-9513.
DT Article
LA English
AB This study describes the properties of a clone of immortalized cells (m-IC-cl2 cells) derived from the bases of small intestinal villi from 20-day-old fetuses of L-type pyruvate kinase (L-PK)/ TAg1 transgenic ***mice*** . The ***mice*** harbor the simian virus 40 large T antigen under the control of the 5' ***regulatory*** sequence from the L-PK gene. m-IC-cl2 cells expressed nuclear large T antigen, had a prolonged life span, and were nontumorigenic when injected into nude ***mice*** . They formed confluent monolayers of cuboid cells separated by tight junctions, developed dense, short apical microvilli, and formed domes. They also possessed cytokeratins, ***villin*** , aminopeptidase N, dipeptidyl-peptidase IV, and glucoamylase and retained crypt cell features, including intracellular sucrase isomaltase and alpha-L-fucose glycoconjugates accumulation and expression of the polymeric immunoglobulin receptor and the cystic fibrosis transmembrane conductance ***regulator*** gene. Thus the mIC-cl2 cell line obtained by targeted oncogenesis in transgenic ***mice*** maintained in culture several important properties and differentiated functions of intestinal crypt cells.

L4 ANSWER 15 OF 22 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.DUPLICATE 12
AN 93123462 EMBASE
DN 1993123462
TI Establishment of renal proximal tubule cell lines by targeted oncogenesis in transgenic mice using the L-pyruvate kinase-SV40 (T) antigen hybrid gene.
AU Cartier N.; Lacave R.; Vallet V.; Hagege J.; Hellio R.; Robine S.; Pringault E.; Cluzeaud F.; Briand P.; Kahn A.; Vandewalle A.
CS Unite 246 INSERM, UER Xavier Bichat, 16 rue Henri Huchard,75018 Paris, France
SO Journal of Cell Science, (1993) 104/3 (695-704). ISSN: 0021-9533 CODEN: JNCSAI
CY United Kingdom
DT Journal; Article
FS 001 Anatomy, Anthropology, Embryology and Histology
004 Microbiology
016 Cancer
028 Urology and Nephrology
029 Clinical Biochemistry
LA English
SL English
AB Targeted oncogenesis allowed us to obtain two cell lines which have been derived from the proximal tubule of kidney from transgenic ***mice*** harbouring the simian virus (SV40) large T and small t antigens placed under the control of the 5' ***regulatory*** sequence from the rat L-type pyruvate kinase (L-PK) gene. The cell lines (PKSV-PCT and PKSV-PR cells) were derived from early (PCT) and late (Pars Recta, PR) microdissected proximal tubules grown in D-glucose-enriched medium. In such conditions of culture, both cell lines exhibited L-PK transcripts, a stable expression of SV40-encoded nuclear large T antigen, a prolonged life span but failed to induce tumors when injected sub-cutaneously into athymic (nu-nu) ***mice*** . Confluent cells, grown on plastic support or porous filters, were organized as monolayers of polarized cuboid cells with well developed apical microvilli and formed domes. Both cell lines exhibited morphological features of proximal tubule cells with ***villin*** located in the apical brush-border and subtantial amounts of hydrolase activity. By immunofluorescence studies using specific antibodies, aminopeptidase N appeared restricted to the apical microvillar domain, whereas the H2 histocompatibility antigen was distributed in the cytoplasm and lateral membranes. These results demonstrate that the proximal morphological phenotype has been fully preserved in these cultured cells derived from tissue-specific targeted oncogenesis in transgenic ***mice*** .

L4 ANSWER 16 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1993:322176 BIOSIS
DN PREV199396030526
TI Establishment of renal proximal tubule cell lines by targeted oncogenesis in transgenic mice using the L-pyruvate kinase-SV40 (T) antigen hybrid gene.
AU Cartier, N.; Lacave, R.; Vallet, V.; Hagege, J.; Hellio, R.; Robine, S.; Pringault, E.; Cluzeaud, F.; Briand, P.; Kahn, A.; Vandewalle, A. (1)
CS (1) Unite 246 INSERM, UER Xavier Bichat, 16 rue Henri Huchard, 75018 Paris France
SO Journal of Cell Science, (1993) Vol. 21, No. 4, pp. 695-704. ISSN: 0021-9533.
DT Article
LA English
AB Targeted oncogenesis allowed us to obtain two cell lines which have been derived from the proximal tubule of kidney from transgenic ***mice*** harbouring the simian virus (SV40) large T and small t antigens placed under the control of the 5' ***regulatory*** sequence from the rat L-type pyruvate kinase (L-PK) gene. The cell lines (PKSV-PCT and PKSV-PR cells) were derived from early (PCT) and late (Pars Recta, PR)

microdissected proximal tubules grown in D-glucose-enriched medium. In such conditions of culture, both cell lines exhibited L-PK transcripts, a stable expression of SV40-encoded nuclear large T antigen, a prolonged life span but failed to induce tumors when injected sub-cutaneously into athymic (nu-nu) ***mice*** . Confluent cells, grown on plastic support or porous filters, were organized as monolayers of polarized cuboid cells with well developed apical microvilli and formed domes. Both cell lines exhibited morphological features of proximal tubule cells with ***villin*** located in the apical brush-border and substantial amounts of hydrolase activity. By immunofluorescence studies using specific antibodies, aminopeptidase N appeared restricted to the apical microvillar domain, whereas the H2 histocompatibility antigen was distributed in the cytoplasm and lateral membranes. These results demonstrate that the proximal morphological phenotype has been fully preserved in these cultured cells derived from tissue-specific targeted oncogenesis in transgenic ***mice*** .

L4 ANSWER 17 OF 22 CAPLUS COPYRIGHT 2003 ACS
AN 1992:627787 CAPLUS
DN 117:227787
TI Villin gene promoter sequence and its use for preparation of transgenic animal models of human tumor growth and proliferation
IN Pringault, Eric; Robine, Sylvie; Huet, Christian; Babinet, Charles; Louvard, Charles
PA Institut Pasteur, Fr.; Institut National de la Sante et de la Recherche Medicale (INSERM)
SO Eur. Pat. Appl., 63 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 496174	A1	19920729	EP 1991-402887	19911028
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
CA 2054149	AA	19920430	CA 1991-2054149	19911024
JP 07008281	A2	19950113	JP 1991-347614	19911029
PRAI US 1990-604905		19901029		

AB The ***promoter*** sequence of human ***villin*** gene is given. It is useful for driving the expression of oncogenes such as ras in mammalian cells and prepn. of transgenic mammals such as ***mouse*** harboring an oncogene of interest. The restriction map of the ***villin*** gene ***promoter*** was given.

L4 ANSWER 18 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE
13
AN 1992:454422 BIOSIS
DN BA94:95822
TI DEVELOPMENTAL ***REGULATION*** OF ***VILLIN*** GENE EXPRESSION IN
THE EPITHELIAL CELL LINEAGES OF ***MOUSE*** DIGESTIVE AND UROGENITAL
TRACTS.
AU MAUNOURY R; ROBINE S; PRINGAULT E; LEONARD N; GAILLARD J A; LOUVARD D
CS INST. PASTEUR, DEPARTEMENT DE BIOLOGIE MOLECULAIRE, URA-CNRS 1149, 25 RUE
DE DOCTEUR ROUX, 75724 PARIS CEDEX 15, FRANCE.
SO DEVELOPMENT (CAMB), (1992) 115 (3), 717-728.
CODEN: DEVPED. ISSN: 0950-1991.
FS BA; OLD
LA English
AB The expression of ***villin*** , an actin-binding protein and major component of the brush border of specialized structural absorptive cells, was studied during ***mouse*** embryogenesis. We show that the ontogeny of ***villin*** expression is limited to the epithelial cell lineages of the digestive and uro-genital tracts and accounts for the tissue-specific expression observed in adult ***mice*** . This spatiotemporal pattern of ***villin*** expression is distinctive in sequence, intensity, regional distribution and polarization. During the development of the primitive gut, ***villin*** is faintly and discontinuously expressed in the invaginating foregut but it is expressed in every cell bordering the hindgut pocket. Later, ***villin*** expression increases along the developing intestine and concentrates in the brush border of the epithelium bordering the villi. In gut derivatives, ***villin*** is present in liver and pancreas primordia but only biliary and pancreatic cells maintain a faint ***villin*** expression as observed in adults. In the urogenital tract, mesonephric tubules are the first mesodermal derived structures to express ***villin*** . This expression is maintained in the ductuli efferentes, paradidymis and epoophoron. ***Villin*** appears in the proximal metanephric tubules and later increases and concentrates in the brush border of the renal proximal tubular epithelial cells. Thus ***villin*** expression can be considered as an early marker of the endodermal cell lineage during the development of the digestive system. Conversely, during the development of the excretory and genital system, ***villin*** is only expressed after the mesenchyme/epithelium conversion following the appearance of tubular structures. These observations emphasize the multiple levels of ***regulation*** of ***villin*** gene activity that occur during ***mouse*** embryogenesis and account for the strict pattern of tissue-specific expression observed in adults. In the future, ***regulatory*** ***elements*** of the ***villin*** gene may be used to target the early expression of oncogenes to the digestive and urogenital tracts of transgenic ***mice*** .

L4 ANSWER 19 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE
14
AN 1992:389517 BIOSIS
DN BA94:61692
TI EXPRESSION AND LOCALIZATION OF VILLIN FIMBRIN AND MYOSIN I IN DIFFERENTIATING MOUSE F9 TERATOCARCINOMA CELLS.
AU EZZELL R M; LEUNG J; COLLINS K; CHAFEL M M; CARDOZO T J; MATSUDAIRA P T
CS SURGERY RESEARCH LABORATORY, MASSACHUSETTS GENERAL HOSPITAL, 149 13TH
STREET, CHARLESTOWN, MASS. 02129.
SO DEV BIOL, (1992) 151 (2), 575-585.
CODEN: DEBIAO. ISSN: 0012-1606.
FS BA; OLD
LA English
AB F9 embryonic carcinoma cells are a multipotent cell line which can be induced to differentiate into cells resembling the visceral endoderm, an extraembryonic absorptive epithelium characterized by apical microvilli. We have examined the role of ***villin*** , fimbrin, and myosin I, the major actin-binding proteins in the intestinal and visceral yolk sac microvilli, in the development of epithelial polarity and the assembly of the microvillus cytoskeleton in differentiating F9 cells. By immunoblot analysis ***villin*** was first detected at 4 days of differentiation. Cofocal microscopy localized ***villin*** at Day 4 to the apical surface and by Day 6 to the basolateral surfaces as well. In comparison, fimbrin and myosin I were both present in undifferentiated F9 cells and became associated with the apical surface after ***villin*** during differentiation to visceral endoderm. The accumulation of ***villin*** , fimbrin, and myosin I at the apical surface in differentiating F9 cells correlated with the appearance of microvilli containing organized actin filament bundles. Two ***mouse*** ***villin*** cDNAs were isolated and characterized to examine ***villin*** expression during F9 differentiation. ***Mouse*** ***villin*** as encoded by two transcripts (3.8 and 3.4 kb) which differ in their 3'-noncoding region. Both ***villin*** mRNAs were first detected by Day 4 of differentiation and their appearance coincided with expression of the visceral endoderm marker .alpha.-fetoprotein. The pattern of expression and order of accumulation of ***villin*** , fimbrin, and myosin I in differentiating F9 cells are common to developing gut and yolk sac epithelium. This suggests that microvillus assembly is directed by a sequence of temporally and spatially ***regulated*** localizations of these actin-binding proteins.

L4 ANSWER 20 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE
15
AN 1992:96094 BIOSIS
DN BA93:52644
TI STRUCTURE OF THE HUMAN VILLIN GENE.
AU PRINGAULT E; ROBINE S; LOUVARD D
CS UNITE BIOLOGIE MEMBRANES, CENTRE NATIONAL RECHERCHE SCIENTIFIQUE, UNITE
ASSOCIEE 1149, DEP. BIOLOGIE MOLECULAIRE, INSTITUT PASTEUR, 25 RUE DU DR.
ROUX, 75015 PARIS, FR.
SO PROC NATL ACAD SCI U S A, (1991) 88 (23), 10811-10815.
CODEN: PNASA6. ISSN: 0027-8424.
FS BA; OLD
LA English
AB We have isolated and characterized the complete human ***villin*** gene. The ***villin*** gene is located on chromosome 2q35-36 in humans on chromosome 1 in ***mice*** . ***Villin*** belongs to a family of calcium- ***regulated*** actin-binding protein that share structural and functional homologies. The ***villin*** gene is expressed mainly in cells that develop a brush border, such as mucosal cells of the small and large intestine and epithelial cells of the kidney proximal tubules. ***Villin*** gene expression is strictly ***regulated*** during adult life and embryonic development in the digestive and urogenital tracts and, thus, may be used as a marker of the digestive and renal cell lineages. The human ***villin*** gene has one copy per haploid genome, encompasses about 25 kilobases, and contains 19 exons. Analysis of the structural organization of this gene shows that the two mRNAs that encode ***villin*** in humans arise by alternative choice of one of the two polyadenylylation signals located within the last exon. The overall organization of the exons reflects the gene duplication event from which this family of actin-binding proteins originated.

L4 ANSWER 21 OF 22 CAPLUS COPYRIGHT 2003 ACS
AN 1992:168389 CAPLUS
DN 116:168389
TI From the structure to the function of villin, an actin-binding protein of the brush border
AU Friederich, Evelync; Pringault, Eric; Arpin, Monique; Louvard, Daniel
CS Dep. Biol. Mol., Inst. Pasteur, Paris, 75724, Fr.
SO BioEssays (1990), 12(9), 403-8
CODEN: BIOEEJ; ISSN: 0265-9247
DT Journal; General Review
LA English
AB A review, with 32 refs. ***Villin*** , a calcium- ***regulated***

actin-binding protein, modulates the structure and assembly of actin filaments in vitro. It is organized into three domains, the first two of which are homologous. ***Villin*** is mainly produced in epithelial cells that develop a brush border and which are responsible for nutrient uptake. Expression of the ***villin*** structural gene is precisely ***regulated*** during ***mouse*** embryogenesis and is restricted in adults, to certain epithelia of the gastrointestinal and urogenital tracts. The function of ***villin*** has been assessed by transfecting CV1 cells with a human cDNA encoding wild-type ***villin*** or mutant ***villin*** . Synthesis of large amts. of ***villin*** in cells which do not normally produce this protein induces the growth of microvilli on the cell surface and the redistribution of F-actin, concomitant with the disappearance of stress fibers. The complete ***villin*** sequence is required for the morphogenic effect. These results suggest that villin plays a key role in the morphogenesis of microvilli.

L4 ANSWER 22 OF 22 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1989:91752 BIOSIS
DN BA87:45888
TI VILLIN EXPRESSION IN THE VISCERAL ENDODERM AND IN THE GUT ANLAGE DURING
EARLY MOUSE EMBRYOGENESIS.
AU MAUNOURY R; ROBINE S; PRINGAULT E; HUET C; GUENET J L; GAILLARD J A;
LOUVARD D
CS LAB. D'ANAT. PATHOL., HOP. SAINTE ANNE, 1 RUE CABANIS, 75674 PARIS CEDEX
14, FR.
SO EMBO (EUR MOL BIOL ORGAN) J, (1988) 7 (11), 3321-3330.
CODEN: EMJODG. ISSN: 0261-4189.
FS BA; OLD
LA English
AB ***Villin*** is an evolutionarily well conserved, Ca2+ ***regulated*** actin-binding protein, and a major structural component of the brush border of specialized absorptive cells. Using paraffin sections and an affinity purified polyclonal anti- ***villin*** antibody, we have investigated the early expression of ***villin*** during ***mouse*** embryogenesis. ***Villin*** is first detectable at the early post-implantation stage in visceral endodermal cells at the periphery of the egg cylinder. In this extra embryonic layer, the expression of ***villin*** increases and then persists until full term gestation. In the embryo, ***villin*** first appears in gut anlage during the axial rotation. Using the same methodology, ***villin*** expression is also demonstrated in differentiating embryoid bodies from a teratocarcinoma. Both in extra embryonic and embryonic extracts, ***villin*** expression is confirmed by immunoblot and Northern blot analysis which reveal, respectively, a single polypeptide of 93 kd and an mRNA of 3.4 kb in length, two well defined parameters for adult ***mouse*** ***villin*** gene expression. The results presented here show that paraffin sections allow very sensitive and highly resolutive detection of antigens in early embryogenesis. They provide a detailed developmental profile of ***villin*** expression and demonstrate the usefulness of ***villin*** as a marker for epithelial cells involved in absorptive processes.

=> d his

(FILE 'HOME' ENTERED AT 17:41:42 ON 16 JAN 2003)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 17:41:51 ON 16 JAN 2003
L1 1347 S VILLIN
L2 164 S L1 (3S) (MOUSE OR MICE OR MURINE)
L3 48 S L2 (3S) (PROMOTER OR 5 UTR OR REGULAT? ELEMENT? OR CIS ELEMEN
L4 22 DUP REM L3 (26 DUPLICATES REMOVED)

=>

---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

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FULL ESTIMATED COST		81.41	81.62

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	ENTRY	SESSION	SINCE FILE TOTAL
CA SUBSCRIBER PRICE		-1.95	-1.95

STN INTERNATIONAL LOGOFF AT 17:53:40 ON 16 JAN 2003